Ginseng: a potential cause of long QT

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Abstract
Ginseng is a frequently used food additive and considered to be relatively safe. Long QT syndrome can be hereditary or acquired. It presents as syncope, sudden cardiac death, or seizures. We report the novel case of a female patient without cardiovascular risk factors who developed prolonged QT with subsequent torsades de pointes during periods in which she was drinking large amounts of ginseng.

Keywords: Ginseng; Long QT; Torsades de pointes

Case report

A 43-year-old healthy woman presented twice to the emergency department for loss of consciousness with brief tonic clonic movements. Result of physical examination on presentation was normal. Episodes recurred in the emergency department and were associated with cyanosis requiring intubation. Electrocardiogram on admission showed a sinus rhythm at 68 beats per minute and corrected QT (QTc) of 720 milliseconds (Bazett formula) (Fig. 1A). Electrolytes’ levels, comprehensive toxicology screening, and neurologic workup had negative results. Seizure was treated with levetiracetam. Corrected QT on discharge was 440 milliseconds. After 3 months, similar syncope recurred. Corrected QT was 645 milliseconds. Levetiracetam level was therapeutic. Monitoring during a second syncopal episode recorded polymorphic ventricular tachycardia (torsades de pointes) (Fig. 1B) treated with magnesium sulfate and lidocaine. Mexiletine was started. Corrected QT normalized to 400 milliseconds. Laboratory workup was normal. Genetic testing was negative for long QT mutations. Patient later admitted consuming since 6 months at least 70 cL of caffeine and 4 L of Korean Panax ginseng daily. Patient was advised to stop ginseng consumption. No subsequent events were noted.

Discussion

In our case, the diagnosis of long QT syndrome was delayed for 3 months as seizure disorder was first erroneously considered. The average delay in establishing long QT syndrome in patients who present with seizure can reach 11.8 years even in the presence of long QT interval that is usually disregarded or miscalculated. In this case, the delayed age of onset of torsades, the absence of familial history of sudden cardiac death, and the negative genetic workup favor the diagnosis of an acquired long QT although a negative genetic testing does not entirely eliminate the diagnosis of congenital long QT syndrome.

The most apparent cause of QT prolongation in the absence of electrolyte disturbances and other drugs that could account for the QT prolongation was the consumption of large quantities of ginseng. In addition, QT used to normalize during periods of abstinence from ginseng.

Ginseng is renowned for its adaptogenic effect mediated by structures called ginsenosides. Experiments have shown that ginsenosides have a prolonging effect on QT. In one animal study, infusion of Rg1 ginsenoside prolonged the ventricular effective refractory period. In another human study, the intake of 250 mg of ginseng by healthy adults increased QTc by 0.015 seconds, a statistically but not clinically significant result. It is not proven whether a higher dose of ginseng or a synergistic effect of caffeine could further prolong QT leading to malignant dysrhythmias. Female sex could have also constituted a favorable setting that facilitates QT prolongation. In conclusion, this is the first case to suspect ginseng to cause torsades de pointes, an effect that still
needs to be proven by further in vitro and animal studies that target its specific arrhythmogenic effect.

References

Fig. 1. (A) Electrocardiogram on admission showing QTc of 720 milliseconds with bifid T waves. (B) Torsades de pointes.